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EXAMINER	
PRIEBE, SCOTT DAVID	
ART UNIT	PAPER NUMBER
1632	

DATE MAILED: 05/09/2002

Please find below and/or attached an Office communication concerning this application or proceeding.

Office Action Summary	Application No.	Applicant(s)	
	09/357,675	CROCE, CARLO M.	
	Examiner	Art Unit	
	Scott Priebe	1632	

-- The MAILING DATE of this communication appears on the cover sheet with the correspondence address --

Period for Reply

A SHORTENED STATUTORY PERIOD FOR REPLY IS SET TO EXPIRE 3 MONTH(S) FROM THE MAILING DATE OF THIS COMMUNICATION.

- Extensions of time may be available under the provisions of 37 CFR 1.136(a). In no event, however, may a reply be timely filed after SIX (6) MONTHS from the mailing date of this communication.
- If the period for reply specified above is less than thirty (30) days, a reply within the statutory minimum of thirty (30) days will be considered timely.
- If NO period for reply is specified above, the maximum statutory period will apply and will expire SIX (6) MONTHS from the mailing date of this communication.
- Failure to reply within the set or extended period for reply will, by statute, cause the application to become ABANDONED (35 U.S.C. § 133).
- Any reply received by the Office later than three months after the mailing date of this communication, even if timely filed, may reduce any earned patent term adjustment. See 37 CFR 1.704(b).

Status

1)☒ Responsive to communication(s) filed on *04 March 2002* .

2a)☐ This action is **FINAL**. 2b)☒ This action is non-final.

3)☐ Since this application is in condition for allowance except for formal matters, prosecution as to the merits is closed in accordance with the practice under *Ex parte Quayle*, 1935 C.D. 11, 453 O.G. 213.

Disposition of Claims

4)☒ Claim(s) *1-3 and 10-12* is/are pending in the application.

4a) Of the above claim(s) *12* is/are withdrawn from consideration.

5)☐ Claim(s) _____ is/are allowed.

6)☒ Claim(s) *1-3,10 and 11* is/are rejected.

7)☐ Claim(s) _____ is/are objected to.

8)☐ Claim(s) _____ are subject to restriction and/or election requirement.

Application Papers

9)☒ The specification is objected to by the Examiner.

10)☐ The drawing(s) filed on _____ is/are: a)☐ accepted or b)☐ objected to by the Examiner.

Applicant may not request that any objection to the drawing(s) be held in abeyance. See 37 CFR 1.85(a).

11)☐ The proposed drawing correction filed on _____ is: a)☐ approved b)☐ disapproved by the Examiner.

If approved, corrected drawings are required in reply to this Office action.

12)☐ The oath or declaration is objected to by the Examiner.

Priority under 35 U.S.C. §§ 119 and 120

13)☐ Acknowledgment is made of a claim for foreign priority under 35 U.S.C. § 119(a)-(d) or (f).

a)☐ All b)☐ Some * c)☐ None of:

1.☐ Certified copies of the priority documents have been received.

2.☐ Certified copies of the priority documents have been received in Application No. _____ .

3.☐ Copies of the certified copies of the priority documents have been received in this National Stage application from the International Bureau (PCT Rule 17.2(a)).

* See the attached detailed Office action for a list of the certified copies not received.

14)☐ Acknowledgment is made of a claim for domestic priority under 35 U.S.C. § 119(e) (to a provisional application).

a) ☐ The translation of the foreign language provisional application has been received.

15)☐ Acknowledgment is made of a claim for domestic priority under 35 U.S.C. §§ 120 and/or 121.

Attachment(s)

1) ☐ Notice of References Cited (PTO-892)
2) ☐ Notice of Draftsperson's Patent Drawing Review (PTO-948)
3) ☐ Information Disclosure Statement(s) (PTO-1449) Paper No(s) _____ .
4) ☐ Interview Summary (PTO-413) Paper No(s). _____ .
5) ☐ Notice of Informal Patent Application (PTO-152)
6) ☐ Other:

DETAILED ACTION

The Group and/or Art Unit designation of your application in the PTO has changed. To aid in correlating any papers for this application, all further correspondence regarding this application should be directed to Primary Examiner Scott D. Priebe, Ph.D., Group Art Unit 1632.

The amendments filed 4/4/01, 8/21/01, 1/22/02 and 3/4/02 have been entered. Claims 13-16 have been cancelled (amendment filed 4/4/01). Claim 11 has been amended (amendment filed 8/29/01). Claims 1-3 and 10-12 are pending.

The text of those sections of Title 35, U.S. Code not included in this action can be found in a prior Office action.

Election/Restriction

Claim 12 is withdrawn from further consideration pursuant to 37 CFR 1.142(b) as being drawn to a nonelected invention, there being no allowable generic or linking claim. Election was made **without** traverse in Paper No. 6, filed 7/3/00..

Specification

The amendment filed 8/21/01 is objected to under 35 U.S.C. 132 because it introduces new matter into the disclosure. 35 U.S.C. 132 states that no amendment shall introduce new matter into the disclosure of the invention. The added material which is not supported by the

original disclosure is as follows: The amendment to the paragraph starting at page 6, line 12 includes identification of GenBank accession numbers "AF069984-AF069989" in the last line of the paragraph. The original specification does not support this added subject matter. The specification was originally filed with the disclosed nucleotide and amino acid sequences listed in the Sequence Listing filed 3/4/02. None of these are identical to the newly recited GenBank accession numbers. The original specification indicates that nucleotides sequences of a human NIT1 gene, human NIT1 cDNA, mouse NIT1 gene, mouse NIT1 cDNA, *D. melanogaster* Nit-Fhit cDNA and *C. elegans* Nit-Fhit cDNA had been submitted to GenBank, but the accession numbers were not provided. No evidence has been made of record that the sequences submitted to GenBank referred to in the specification are identical to those of Acc. Nos. AF069984-AF069989, nor does the specification provide any indication that the GenBank sequences to be incorporated by reference into the specification.

Furthermore, since the sequences were not included with the original specification, there is no evidence of record that Applicant had contemplated the sequence information as being part of the original disclosure or invention. According to the GenBank records, the sequences were submitted to GenBank on 6/4/98, well before provisional application 60/093,350 was filed on 7/20/98, and were published on 7/23/98, almost a year before the filing date of the instant application. Thus, had Applicant intended the sequences to be part of the provisional application, the sequences could have and should have been included; and had Applicant intended the

sequences to be part of the instant application, the sequences could have and should have been included.

Applicant is required to cancel the new matter in the reply to this Office action.

The disclosure is objected to because it contains embedded hyperlink and/or other form of browser-executable code. Applicant is required to delete all embedded hyperlinks and/or other forms of browser-executable code. See MPEP § 608.01. See page 13, line 4, for example. Applicant should review the specification for other embedded hyperlinks and/or other form of browser-executable code.

The disclosure is objected to because of the following informalities: the brief description for Figure 6 does not clearly relate to what is shown in Fig. 6. According to the Sequence Listing, SEQ ID NO: 1 shown in Fig. 6 is a human DNA of some kind labeled NITD. It clearly is not a genomic DNA as it clearly lacks intron sequences as indicated in Fig. 3. A sequence comparison of SEQ ID NO: 25, shown in Fig. 6, to SEQ ID NOs: 21-24 reveals that the amino acid sequence from residue number 37 to 362 matches nucleotides 2-323 of the human NIT1 protein of SEQ ID NO: 21 (and Fig. 1), but the first 36 amino acids do not match. The specification does not otherwise discuss what Figure 6 is supposed to represent, but goes so far as to distinguish it from nucleic acids encoding a NIT1 protein (compare page 4, lines 14-16 with lines 4-13).

Appropriate correction is required.

Claim Rejections - 35 USC § 112

Claims 1-3, 10 and 11 are newly rejected under 35 U.S.C. 112, first paragraph, as containing subject matter which was not described in the specification in such a way as to reasonably convey to one skilled in the relevant art that the inventor(s), at the time the application was filed, had possession of the claimed invention.

Claims 1-3, 10 and 11 are rejected as being based upon a specification into which new matter has been introduced, as discussed above, in a manner which affects the written description of the claimed invention. The introduction of the GenBank accession numbers into the specification introduces species which are readable on the claims. These species were not described in the specification as originally filed, and when read in light of the amended specification, the scope of the instant claims is materially affected.

Claims 1-3, 10 and 11 are newly rejected under 35 U.S.C. 112, first paragraph, as containing subject matter which was not described in the specification in such a way as to enable one skilled in the art to which it pertains, or with which it is most nearly connected, to make and/or use the invention.

The claims are directed to any purified *NIT1* gene wherein said gene is a human gene or mammalian gene (Claims 1-3). The claims are further directed to any isolated nucleic acid of less than 100 kb comprising a nucleotide sequence encoding a Nit1 protein wherein said protein is a human Nit1 protein (claims 10-11). The *only* disclosed uses of the claimed invention are

for treating or preventing an unspecified disease or disorder in a subject or for diagnosing or screening for the presence of or a predisposition for developing an unspecified disease or disorder in a subject comprising detecting one or more mutations in *NIT1* DNA, RNA or Nit1 protein derived from the subject.

The specification does not contain any guidance whatsoever as to how the claimed invention should be used either to treat or to diagnose any disease, nor does it even disclose what disease(s) could be treated or diagnosed. There are no working examples of using the claimed invention for either treatment or diagnosis. There is no evidence presented in the specification that a change in NIT1 function or expression is related to any disease. Consequently, it is completely unpredictable what disease(s), if any, the invention could be used to treat or diagnose.

The rule that a specification need not disclose that which is well known in the art simply means that omission of minor details does not cause a specification to fail the enablement requirement, and is not a substitute for an enabling disclosure. However, if there is no disclosure of starting materials and of conditions under which the process can be carried out, undue experimentation is required. Failure to provide such teachings can not be rectified by asserting that the disclosure of the missing necessary information was well known in the prior art. See *Genentech Inc. v. Novo Nordisk A/S*, 42 USPQ2d 101, 1005 (CA FC, 1997). In this case, the prior art is of little or no help at all. Although the concept of gene therapy has potential, the realities of the parameters which will result in therapeutic benefit have not been achieved and are considered unpredictable. With regard to *in vivo* gene transfer, the specification provides no

example or therapeutic methodology. For example, Eck & Wilson (The Pharmacological Basis of Therapeutics, 1996) teach numerous factors complicate the gene delivery art which would not have been shown to be overcome by routine experimentation. These include, the fate of the DNA vector itself (volume of distribution, rate of clearance into the tissues, etc.), the *in vivo* consequences of altered gene expression and protein function, the fraction of vector taken up by the target cell population, the trafficking of the genetic material within cellular organelles, the rate of degradation of the DNA, the level of mRNA produced, the stability of the mRNA produced, the amount and stability of the protein produced, and the protein's compartmentalization within the cell, or its secretory fate, once produced. These factors differ dramatically based on the vector used and the protein being produced, which cells are target cells, and the disease and/or host being treated. It is further noted that Eck and Wilson support the importance of tailoring a gene therapy vector and method to specific diseases and/or disorders. See page 82, column 1, first paragraph. Furthermore, Eck & Wilson et al. review the state of the art for gene therapy for inherited disorders and discloses that "[t]he level of protein function necessary to achieve complementation of the defect varies widely among genetic diseases." See page 78, column 2, 2nd paragraph.

In addition, while progress has been made in recent years for gene transfer *in vivo*, vector targeting to desired tissues *in vivo* continues to be unpredictable and inefficient as supported by numerous teachings available in the art. For example, Miller (1995, FASEB J., Vol. 9, pages 190-199) review the types of vectors available for *in vivo* gene therapy, and conclude that "for the

long-term success as well as the widespread applicability of human gene therapy, there will have to be advances...targeting strategies outlined in this review, which are currently only at the experimental level, will have to be translated into components of safe and highly efficient delivery systems" (page 198, column 1). Deonarain (1998, Expert Opin. Ther. Pat., Vol. 8, pages 53-69) indicate that one of the biggest problems hampering successful gene therapy is the "ability to target a gene to a significant population of cells and express it at adequate levels for a long enough period of time" (page 53, first paragraph). Deonarain reviews new techniques under experimentation in the art which show promise but states that such techniques are even less efficient than viral gene delivery (see page 65, first paragraph under Conclusion section). Verma (Sept. 1997, Nature, Vol. 389, pages 239-242) reviews vectors known in the art for use in gene therapy and discusses problems associated with each type of vector. The teachings of Verma indicate a resolution to vector targeting has not been achieved in the art (see entire article). Verma also teaches appropriate regulatory elements may improve expression, but it is unpredictable what tissues such regulatory elements target (page 240, sentence bridging columns 2 and 3). Crystal (1995, Science, Vol. 270, page 404-410) also reviews various vectors known in the art and indicates that "among the design hurdles for all vectors are the need to increase the efficiency of gene transfer, to increase target specificity and to enable the transferred gene to be regulated" (page 409).

Specifically, the specification, on page 7, teaches only *in vitro* multiple tissue northern blots of *NIT1* cDNA probes. However, no further results are reported on the effectiveness of

Nit1 protein function having any implication toward the treatment of any particular diseases or disorders. It is noted that, Orkin stress the importance of using relevant animal models for determining the effectiveness of therapeutic methodologies (p. 10 and 13). As such, the specification fails to provide any evidence which would provide a reasonable nexus to that of any particular diseases or disorders.

With regard to a method of diagnosing or screening for the presence of or a predisposition for developing a disease or disorder in a subject comprising detecting one or more mutations in *NIT1* DNA or RNA derived from the subject in which the presence of said one or more mutations indicates the presence of the disease or disorder or a predisposition for developing the disease or disorder, the specification fails to teach or suggest any methodology or procedure for a method of diagnosing or screening of any mutations in *NIT1* DNA or RNA correlating to any disease or disorder in any subject as embraced by the claim. The specification only discloses that the pattern of *Nit1* expression was the same in different human cells types, and differed between some mouse cell types, and in mice, was almost identical to the pattern of the expression of Fhit. The specification suggests that the expression pattern in mice support the hypothesis that the NIT1 proteins may act in concert or participate in the same pathway as FHIT (page 14, lines 1-4), but does not teach what that pathway is. More important, the specification fails to discuss any methods of screening or diagnosing of any disease or disorder in any subject comprising detecting any mutations in *NIT1* DNA or RNA in which the presence of any said mutations would indicate the presence of any disease or disorder. The specification further fails to indicate

that any mutations in *NIT1* DNA or RNA would even correlate to any disease or disorder. Thus, it would be unpredictable for one of skill in the art to identify mutations of *NIT1* DNA or RNA which would result in any disease or disorder as embraced by the claimed invention.

Accordingly, in view of the unpredictable and undeveloped state of the art, the lack of guidance or working examples which demonstrate or correlate to any therapeutic effect of the claimed methods, including the identification of any Nit1 mutations, and the breadth of the claims, the specification fails to teach any nucleic acid sequences which "enhance or inhibit" a Nit 1 protein as embraced by the claims directed to a method of treating or preventing any disease or disorder in any subject, including any are disease relevant Nit 1 mutations.

There is no evidence of record for any well established use of the claimed invention. The specification does not disclose either a biochemical or biological function for a NIT1 gene or protein. Unknown hypothetical relationships to FHIT function do not suggest any way to use the claimed NIT1 nucleic acids. While the specification teaches that the mammalian NIT1 genes are homologous to plant and bacterial nitrilase genes, it does not teach or suggest that the mammalian NIT1 proteins are nitrilases, or if they were, what nitriles they act on. Applicant has stated on the record (amendment filed 3/27/01) that the NIT1 of the claimed invention "are distinct from the nitrilases", which indicates that the mammalian NIT1 is not a nitrilase. The invention could conceivably be used to determine the biological or biochemical function or activity of NIT1 genes or proteins, and to determine what, if any, relationship the mammalian NIT1 proteins have with FHIT. However, such use amounts to using the invention to further

study or characterize itself. Such use does not meet the requirements of §101 or §112, first para. In *Brenner v. Manson*, 148 USPQ 689, 696 (US SupCt., 1966), the Supreme Court noted that "Congress intended that no patent be granted on a chemical compound whose sole "utility" consists of its potential role as an object of use-testing", and stated, in context of the utility requirement, that "a patent is not a hunting license. It is not a reward for the search, but compensation for its successful conclusion."

Claims 1-3 and 10 remain rejected under 35 U.S.C. 112, first paragraph, for the reasons of record set forth in the Office action of 9/27/00 as containing subject matter which was not described in the specification in such a way as to reasonably convey to one skilled in the relevant art that the inventor(s), at the time the application was filed, had possession of the claimed invention.

Applicant's arguments filed 3/27/01 have been fully considered but they are not persuasive. While the description beginning on page 12 of the specification indicates that Applicant had isolated a NIT1 gene from mouse and human, and determined their nucleotide sequences, the original disclosure did not provide the nucleotide sequences. The cartoon in Fig. 3A shows approximate sizes of exons and introns, but this information does not convey the sequence of the "gene". The nucleotide sequence shown in Fig. 6 is not disclosed as being a gene sequence - a gene sequence is a type of genomic sequence. The amino acid sequences shown in Figure 1 shows the amino acid sequences for the mouse and human NIT1 proteins and compares

them to Nit-Fhit proteins (*not* NIT1 proteins) of two invertebrates. The amino acid sequences describe what the mouse and human genes encode, but do not allow one to determine either the nucleotide sequence which encodes these amino acids or the non-coding sequences, such as introns, included in the mouse and human "gene".

Claims 1 and 2 are not only directed to mouse or human NIT1 genes, but also to any NIT1 gene from any organism, including plants, bacteria, etc. The rejection of record also raised the issue of whether the specification was adequate to describe a genus of NIT1 genes or nucleic acid encoding a NIT1 protein, based only on disclosure of the human and mouse NIT1 proteins and their sequence similarity to Nit-Fhit proteins of invertebrates, which are not NIT1 proteins. The only identifying characteristics for the mouse and human genes are the putative amino acid sequences they encode (Fig. 1) and the arrangement and spacing of the exons and introns (Fig. 3) and their approximate chromosomal location. These identifying characteristics for any other NIT1 gene or protein are not described, whether from mammals or any other organism. The specification does not describe characteristics of a NIT1 gene or protein that would distinguish the claimed nucleic acid from any other, or a mammalian NIT1 gene or protein that would distinguish it from any NIT1 gene from a non-mammal. Applicant's response does not address these broader issues in the context of written description.

Applicant's traversal of the rejection under §102 illustrates the inadequacy of the written description of the claimed inventions in the disclosure. The specification does not define "NIT1 gene" or "NIT1 protein". The NIT1 proteins of human and mice are defined by the disclosed

amino acid sequence, and their genes by the amino acid sequences disclosed and the arrangement of their exons and introns. The specification does not describe how this information can be extrapolated to arrive at other species of NIT1 genes or proteins embraced by the claims. The specification teaches that the human and mouse NIT1 genes and proteins are members of an uncharacterized family of mammalian genes and proteins homologous to nitrilases and amidohydrolases of plants and bacteria (page 3). The specification does not teach whether the mouse and human NIT1 proteins are nitrilases or amidohydrolases. In teaching that the mammalian NIT1 genes are uncharacterized as to function, Applicant tacitly admits that the function of the mammalian NIT1 genes and proteins is unknown. Hillebrand et al. teach a gene from *Arabidopsis thaliana* they call *nit1*, which encodes NIT1 protein. This *Arabidopsis* NIT1 protein is homologous to the mouse and human NIT1 protein (page 7 of response). Applicant states that the claimed NIT1 genes are distinct from the plant and bacterial nitrilases. The only support for this statement is that the "*NIT1* genes have a much closer relationship to *FHIT*". However, the specification does not teach what that relationship is, nor how it could be used to distinguish a claimed NIT1 gene from a NIT1 gene excluded from the claims, e.g. the *Arabidopsis nit1* gene. The only relationship disclosed in the specification is that the mouse NIT1 and FHIT genes have almost the same expression pattern in different cell types. However, the specification does not teach that human NIT1 expression parallels human FHIT expression. Given that the biological function of FHIT is unknown and no biochemical or biological function of mammalian NIT1 proteins has been identified, one of skill in the art cannot use this

hypothetical unknown relationship between FHIT and NIT1 to distinguish the claimed genes and nucleic acids from those not being claimed.

The rejection of claim 11 is withdrawn. Figure 1 describes the amino acid sequence of the human NIT1 protein. Given the knowledge in the prior art on the genetic code, one of skill in the art can readily envision nucleic acid that encode the human NIT1 protein.

Claims 1-3, 10 and 11 remain rejected under 35 U.S.C. 112, second paragraph, for the reasons of record set forth in the Office action of 9/27/00 as being indefinite for failing to particularly point out and distinctly claim the subject matter which applicant regards as the invention.

Applicant's arguments filed 3/27/01 have been fully considered but they are not persuasive. Applicant asserts that the NITD sequence of Fig. 6 is NIT1. However, Applicant does not indicate where the specification teaches this. The claims have been rejected primarily because the metes and bounds of "NIT1" are unclear. In response, Applicant states they believe the claims are definite, without providing any argument in support of their position. The specification does not define what NIT1 means. Hildebrand et al. disclose a gene they call *nit1* encoding a nitrilase they call NIT1. Applicant asserts (page 7 of response) that this homologous plant NIT1 and nucleic acid encoding other bacterial is not what they are claiming. However, given the failure to disclose meaningful distinguishing characteristics of claimed NIT1 genes, it is not clear how

Applicant arrives at this conclusion, or more importantly, how one of skill in the art would arrive at this conclusion when reading the claims in light of the specification.

Claim Rejections - 35 USC § 102

Claims 1 and 10 remain rejected under 35 U.S.C. 102(b) as being anticipated by Hillebrand et al (May 8, 1996) Gene, Vol. 170 (2): 197-200 for the reasons of record set forth in the Office action of 9/27/00.

Applicant's arguments filed 3/27/01 have been fully considered but they are not persuasive. In response to applicant's argument that the references fail to show certain features of applicant's invention, it is noted that the features upon which applicant relies (i.e., an unknown, hypothetical relationship between NIT1 and FHIT) are not recited in the rejected claim(s). Although the claims are interpreted in light of the specification, limitations from the specification are not read into the claims. See *In re Van Geuns*, 988 F.2d 1181, 26 USPQ2d 1057 (Fed. Cir. 1993). Given that the specification does not teach what this relationship is, or that it holds for any other organism but mouse, it is not clear how the claims could be amended to avoid this rejection, other than by limiting the claims to mammalian genes or proteins, or to mouse or human genes or proteins. The specification does not define the term NIT1 gene or NIT1 protein, it only provides non-limiting examples of the proteins. Consequently, it must be given its broadest reasonable interpretation. Claims 1 and 10 are directed to nucleic acid comprising *any* NIT1 gene or encoding *any* NIT1 protein, and therefor clearly embrace the Hildebrand nucleic

acid. Indeed, since the only definitive characteristic disclosed for NIT1 is the homology to nitrilases, these claims could be interpreted to read on any homologue of a nitrilase or amidohydrolase gene. The numerical designation "1" is purely historic and arbitrary, and conveys no meaning as to what the claims might embrace.

Certain papers related to this application may be submitted to Art Unit 1632 by facsimile transmission. The FAX numbers are (703) 308-4242 or (703) 305-3014 for any type of communication. In addition, FAX numbers for a computer server system using RightFAX are also available for communications before final rejection, (703) 872-9306, and for communications after final rejection, (703) 872-9307, which will generate a return receipt. The faxing of such papers must conform with the notices published in the Official Gazette, 1156 OG 61 (November 16, 1993) and 1157 OG 94 (December 28, 1993) (see 37 CFR 1.6(d)). NOTE: If applicant *does* submit a paper by FAX, the original copy should be retained by applicant or applicant's representative. NO DUPLICATE COPIES SHOULD BE SUBMITTED, so as to avoid the processing of duplicate papers in the Office.

Any inquiry concerning this communication or earlier communications from the examiner should be directed to Scott D. Priebe whose telephone number is (703) 308-7310. The examiner can normally be reached on Monday through Friday from 8 AM to 4 PM. If attempts to reach the examiner by telephone are unsuccessful, the examiner's supervisor, Deborah Reynolds, can be reached on (703) 305-4051.

Any inquiry concerning administrative, procedural or formal matters relating to this application should be directed to Patent Analyst Patsy Zimmerman whose telephone number is (703) 308-8338. Any inquiry of a general nature or relating to the status of this application should be directed to the Group receptionist whose telephone number is (703) 308-0196.

Scott D. Priebe

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